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Important Books Received

Lectures [6] on the biologic aspects of colloid and physiologic chemistry. A series of lectures given at the Mayo Foundation and the Universities of Minnesota, Iowa, Washington (St. Louis), and the Des Moines Academy of Medicine, Iowa. By six investigators in the field. Introduction by Louis B. Wilson, M.D., Director of the Mayo Foundation for Medical Education and Research. 1927: pp. 244; 6¼ x 3¾ in.; 85 illustrations; $2.50. W. B. Saunders Company, Philadelphia and London.

American illustrated medical dictionary. By W. A. Newman Dorland, A.M., M.D., F.A.C.S., member of the Committee on Nomenclature and Classification of Diseases, American Medical Association, and Editor of the "American Pocket Medical Dictionary;" with the collaboration of E. C. L. Miller, M.D., Professor of Bacteriology and Biochemistry, Medical College of Virginia. *Fourteenth edition.* 1927: pp. 1388; 8¼ x 4¾ in.; 319 illustrations; flexible binding—plain, $7.00; thumb index, $7.50. W. B. Saunders Company, Philadelphia and London.

Dental education in the United States and Canada. By William J. Gies, M.S., Ph.D., Sc.D., LL.D., F.A.C.D., Professor of Biological Chemistry, Columbia University; Executive officer of the Board of Editors, *Journal of Dental Research;* with a preface by Henry S. Pritchett, Ph.D., Sc.D., LL.D., Litt.D., President of the Carnegie Foundation for the Advancement of Teaching. 1926 (issued in 1927): pp. xxii + 692; 7¼ x 5 in.; one illustration; distributed free of charge on application, by letter or in person (the volume is "Bulletin No. Nineteen"). Carnegie Foundation for the Advancement of Teaching, 522 Fifth Avenue, New York City.

Physical chemistry and biophysics, for students of biology and medicine. By Matthew Steel, Ph.D., Professor of Biological Chemistry, Long Island College Hospital. 1928: pp. 372; 7¼ x 4¾ in.; 38 illustrations; $4.00. John Wiley and Sons, Inc., New York City.

Fundamental principles of a systematic diagnosis of dental anomalies, with an explanation of a new classification based upon gnathostatic methods of investigation. By Paul W. Simon, Head of the Department of Orthodontics, Dental Institute, University of Berlin. *Translated* by B. E. Lischer, D.M.D., F.A.C.D., Non-resident Lecturer on Orthodontics, Dental School, University of Michigan. 1926: pp. 378; 6¾ x 4 in.; 189 illustrations; $5.00. Stratford Company, Boston.

The practice of periodontia. By Sidney Sorrin, D.D.S., Chief of Periodontia Clinic, and Samuel C. Miller, D.D.S., Instructor, Periodontia Clinic, College of Dentistry, New York University. 1928: pp. 160; 6 x 3¾ in.; 122 illustrations; $3.50. MacMillan Company, New York.

The romance of the atom. By Benjamin Harrow, Ph.D., Associate in Biological Chemistry, Columbia University. 1927: pp. 162; 5¼ x 3¼ in.; 7 illustrations; $1.50. Boni and Liveright, New York.

The nature of matter, gravitation, and light. By Albert P. Mathews, Ph.D., Professor of Biochemistry, University of Cincinnati. 1927: pp. 222; 6¾ x 4 in.; 7 illustrations; $7.00. William Wood and Co., New York City.

The religion called behaviorism. By Louis Berman, M.D., Associate in Biological Chemistry, Columbia University. 1927: pp. 153; 5¼ x 3¼ in.; no illustrations; $1.75. Boni and Liveright, New York.

Dental state board questions and answers. By R. Max Goepp, M.D., Professor of Principles and Practice of Medicine and of Clinical Medicine, Woman's Medical College of Pennsylvania, Philadelphia. *Fourth edition.* 1928: pp. 463; 7¼ x 4¾ in.; no illustrations; $4.50. W. B. Saunders Co., Philadelphia and London.

American universities and colleges. By David A. Robertson, Assistant Director, American Council on Education, Washington, D. C. 1928: pp. 884; 7¾ x 5¼ in.; no illustrations; $2.50. Charles Scribner's Sons, New York and London.

An introduction to organic chemistry. By Alexander Lowy, Ph.D., Professor of Organic Chemistry, University of Pittsburgh, and Benjamin Harrow, Ph.D., Associate in Biological Chemistry, Columbia University. *Second edition.* 1928: pp. 407; 7 x 4 in.; 22 illustrations; $3.00. John Wiley and Sons, Inc., New York.

A text-book of general bacteriology. By Edwin O. Jordan, Ph.D., Professor of Bacteriology, University of Chicago and Rush Medical College, Chicago, Ill. *Ninth edition.* 1928: pp. 778; 7¼ x 4¾ in.; 191 illustrations; $6.00. W. B. Saunders Co., Philadelphia and London.

Laboratory manual of physiological chemistry. By Meyer Bodansky, Associate Professor of Biological Chemistry, and Marion S. Fay, Adjunct Professor of Biological Chemistry, School of Medicine, University of Texas. 1928: pp. 234; 7¼ x 4¾ in.; 9 illustrations; $2.00. John Wiley and Sons, Inc., New York City.

Bone as a measure of development; when and how we acquired our teeth. By J. S. Foote, A.M., M.D., LL.D., Research Professor in the Dental Department of Creighton University. (*Posthumous.*) Introduction by Arthur D. Black, Dean of Northwestern University Dental School, Chicago. 1928: pp. 187; 8 x 5 in.; 251 illustrations. Scientific Foundation and Research Commission, American Dental Association, Chicago.

Dentition as a measure of maturity. By Psyche Cattell, M.A., Ed.M., Ed.D., Research Associate, Psycho-Educational Clinic, Harvard University, Cambridge, Mass. 1928: pp. 91; 7¼ x 4¾ in.; 36 figures; $1.00. Harvard University Press, Cambridge, Mass.

Four lectures on radiology and radio-therapy. Delivered (in 1927) under the auspices of the Dental Board of the United Kingdom. 1928: pp. 92; 7¼ x 4¾ in.; 109 figures. Dental Board of the United Kingdom, 44 Hallam St., Portland Place, London, W. I.

Treatment of the periodontal pocket. By Harold Keith Box, D.D.S., Ph.D., Research Professor of Periodontology, Faculty of Dentistry, University of Toronto. Foreword by Wallace Seccombe, Dean of the Dental School of the University of Toronto, 1928: pp. 125; 6¾ x 4¾ in.; 120 figures. University of Toronto Press, Toronto, Canada.

Dental histology and embryology. By B. Orban, M.D., Professor of Dental Histology and Pathology, Chicago College of Dental Surgery, Dental Department of Loyola University. 1928: pp. 223; 6¾ x 4 in.; 192 illustrations; $5.00. Rogers Printing Company, Chicago.

Nutrition. By Walter H. Eddy, Ph.D., Professor of Physiological Chemistry, Teachers College, Columbia University, and Associate Director of the Bureau of Foods and Sanitation of "Good Housekeeping Magazine." 1928: pp. 237; 5¼ x 3¼ in.; no illustrations; $2.50. The Williams & Wilkins Company, Baltimore, Md.

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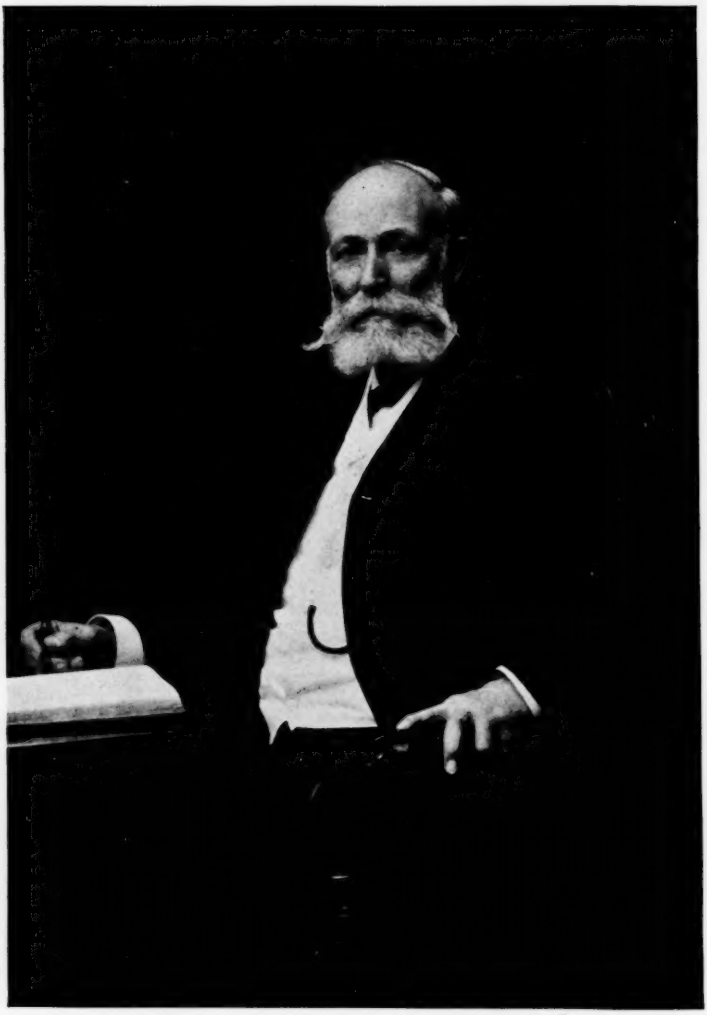

EDWARD AUGUSTUS BOGUE

EDWARD AUGUSTUS BOGUE[1]

WILLIAM B. DUNNING

New York City

EDWARD AUGUSTUS BOGUE[2] was born in Vernon, New York, March 12, 1838, the son of Rev. Horace Publius Bogue and Grace Caroline (Brown) Bogue; and died in New York City, in his eighty-fourth year, November 21, 1921. His mother was of Scotch Presbyterian ancestry. His early education was received at Seneca Falls Academy, New York. The death of an older brother, a dentist, influenced young Bogue in the choice of his profession and he entered the New York College of Dental Surgery at Syracuse, and graduated from that institution with the degree of D.D.S., in 1855. For a time after graduation he was associated with Dr. Amos Westcott of Syracuse—the distinguished pioneer in dental education and dental practice, and father of the writer, in later years, of the famous "David Harum." Following came a short period of practice for himself at Ithaca, New York—when Dr. Bogue moved to Chicago and became associated with Dr. William H. Kennicutt. Later he began an independent practice and remained in Chicago for about eight years.

At this time Dr. Bogue decided upon the study of general medicine, and entered Rush Medical College; but the course was completed at the Medical College of Castleton, Vermont. Upon his return to Chicago he continued practice for a short while, but was then compelled by ill health to give up for a prolonged rest. He spent a year in Switzerland and recovered the splendid vitality which carried him through a long and strenuous career.

In October, 1864, Dr. Bogue began the practice of his profession in

[1] Dr. Bogue was one of the founders of the International Association for Dental Research in 1920.—[*Ed.*]

[2] Besides his personal recollections, in the preparation of this article, the writer is indebted for information to Miss Edith A. Bogue, daughter of the late Dr. Bogue; to Dr. Sebert E. Davenport, for many years associated in practice with Dr. Bogue; to Dr. Henry W. Gillett, and to an obituary notice in the *Dental Cosmos*, vol. lxiv, 1922, p. 253.

New York City—and there he continued for the remainder of his life. He was at first associated with the late Dr. Norman W. Kingsley, who was distinguished by his originality and skill in palatal restorations and in orthodontia. In this office, at 29 East 20th Street, Dr. Bogue built up a thriving practice. In 1870 he became a lecturer in the Harvard University Dental School.

At about this time Dr. Bogue and Dr. Hitchcock, then dean of the Harvard school, evolved a plan to open an office in Paris for work during the summer months. The scheme was deferred because of the death of Dr. Hitchcock, but in 1877 Dr. Bogue associated with him Dr. Cook of Brooklyn, Dr. Moffit of Boston, and Dr. Daboll of Buffalo, and under the partnership name of Bogue, Moffit, Cook and Daboll, an office was established at 39 Boulevard Haussmann, Paris, France. Dr. Isaac B. Davenport was later associated with this group, and during the next quarter century this establishment enjoyed an international reputation. Dr. Bogue personally divided his time about equally between the Paris and New York offices, and so continued for about twenty-three years, when he withdrew from the Paris connection. Until shortly before his death he remained in active practice in New York City, his office for many years having been at 63 West 48th Street.

Dr. Bogue was a conspicuous figure in the progress of dental science in this country and abroad for more than fifty years. During the first half or two thirds of his career he was an active contributor to current dental literature—as the writer of articles and discusser of papers read before societies—and was personally active in the work of dental organizations. He was a charter member and for a time secretary of the American Dental Association, and served as president of the New York Odontological Society, the First District Dental Society of the State of New York, and the New York Institute of Stomatology. He was prominent in the affairs of the New York State Dental Society, the American Academy of Dental Science of Boston, the Odontological Society of Great Britain, the Odontological Society of France, and the International Dental Federation. His official duties included twenty-one years of service on the Board of Dental Examiners of the State of New York.

Dr. Bogue was endowed with a restless and aggressive mentality.

He was widely informed as to general knowledge, kept in touch with current medical progress, cultivated acquaintance with physicians, and approached his dental problems from a broad fundamental viewpoint. In details of operative technique, he was original, inventive, and always a student. In debate he was keen, relentless, not always free from prejudice against individuals, nor, in the minds of his colleagues, always above the suspicion of a love of victory. There was often a hint of satire, in dealing with an opponent who had committed himself, which was disconcerting; but it is the belief of those who knew him at close range that this rather formidable quality was no more than a fault of manner, where only kindliness was meant. He spoke French easily—was familiar with other European languages, and through his professional contacts came to know many prominent and interesting persons.

Dr. Bogue was fond of music, particularly that of the opera, but he had practically no avocations, diversions or hobbies; his profession absorbed his time and strength. One who knew him intimately for thirty-five years has informed the writer of this article that he never knew Dr. Bogue to play a game, or to show any interest in such relaxations. His ability as an operator in all departments of dentistry made him skeptical of the modern tendency to specialization. He was indefatigable in his office work—which was often prolonged by artificial light far into the evening—and he was not always sympathetic with an associate inclined to consider six o'clock time to call a halt. It is said that in going to Paris or arriving in New York, he so arranged as to be at his operating chair within an hour or two after the train arrival or the docking of the steamer.

The care of children's teeth was always a major interest with Dr. Bogue, and in his later years he devoted his time increasingly to the study and practice of orthodontia. When nearly eighty he wrote and published, in a series of articles, an exhaustive study on this subject.

Dr. Bogue's courage in meeting the vicissitudes of life was as remarkable as his endurance of its burdens. In his old age, in a successful effort to check malignant disease, he was forced to submit to the loss of a leg. This calamity did not, however, prevent his attendance upon his patients for several years thereafter, and until a few weeks before the end.

Dr. Bogue was married to Amelia Ingalls in 1866. He was survived by three daughters, Mrs. A. T. Campbell of Montclair, New Jersey, and Misses Edith A. Bogue and Edna L. Bogue. His son, Dr. Frederick L. Bogue, well known as a practitioner and for his services to dental literature, had died about ten years previously.

OBSERVATIONS ON THE RÔLE OF THE ENDOCRINES IN DENTAL DEVELOPMENT

A Clinical Study[1]

WM. G. DOWNS, Jr., M.A., D.D.S.

Department of Anatomy, University of Pennsylvania, Philadelphia, Pennsylvania

In a previous paper (Downs, 1927) the probable lack of correlation between endocrine dystrophies and definite dental anomalies was pointed out. The study upon which that paper was based included both experimental evidence and that offered by over six hundred endocrine clinical cases. Attention was called to the fact that although such symptomatic parallels were lacking, there was observed a very high percentage of dental anomalies of all types, scattered very irregularly throughout the whole group of endocrine cases. This might have been supposed for two reasons: first, because a tremendously high percentage of the endocrine cases are found in degenerating stock, so that any or all of the stigmata of degeneracy may be expected; and second, because any type of developmental interference, nutritive, pathological, or otherwise occurring during the period of gestation may be expected to be in varying degree reflected in many of the tissues or organs at that time undergoing formation. Kaplan (1917) and many others have believed that they could see, in dental anomalies of many kinds, symptomatic evidence of endocrine syndromes. This the writer's earlier study did not support. A more recent one, conducted with the assistance of Dr. George R. Moore of the Department of Orthodontics of the Michigan University School of Dentistry, tends to confirm the earlier study.

In this later study 2800 patients of the Michigan Home and Training School, at Lapeer, Michigan, were examined. As in the previous one, they were first classified in accord with the endocrine diagnosis, and then according to the dental picture presented. No very marked

[1] This study was completed in 1927–28 while the author was a member of the staff of the Department of Anatomy, College of Medicine, University of Michigan.

variations from the findings of the previous study were noted. There was a slight increase in the percentage of prognathisms noted in cretins and acromegalics, and in that of mesognathisms in microcephalics. This difference was due to a considerable improvement in the diagnostic methods used, particularly in having the advice of an able orthodontist in making the dental diagnoses. Other than in these cases, the differences in percentages between the two studies was so slight as to make the publication of the charts evolved from the later study superfluous. For that reason they are dispensed with in this paper, and the reader is referred to the earlier paper for the figures contained therein.

While there is little doubt that the endocrines do influence dental development, just as they do general skeletal growth, this influence is not in the direction of producing definite types of anomalies. The spacing of the anterior teeth noted in many endocrine cases, as well as the mesocclusions frequently seen, are not, apparently, of direct endocrine etiology, but are due rather to the bony overdevelopment of the parts involved. Such an effect may be indirectly of endocrine origin, as pointed out by Petzel (1922) and others, but is more often genetic in origin, as suggested in the earlier study already referred to. Further than this, it would appear to be more than likely that the genetic factor influences the whole syndrome in most of the endocrine cases seen.

In the present study attention was paid to the decay and soft tissue lesions present in the mouths of the patients examined. It was found that a tremendously high percentage of both of these conditions presented. This was true in spite of the fact that the institution has the advantage of the services of a capable full-time dentist, and an oral hygienist as well. Both decay and soft-tissue lesions were present in the mouths of practically one hundred per cent of the cretins, mongols, acromegalics, dwarfs, giants, epileptics and chorea patients, while the percentage was noticeably high in almost all of the glandular cases examined.

The work of Erdheim (1911), Gies (1917 and 1921), Toyofuku (1911) and others has demonstrated the profound influence of the parathyroids upon the teeth. In spite of numerous efforts other workers have failed to present equally convincing arguments for similar

influence by other glands of internal secretion. Hammett (1922) and Appleton (1925) have presented indisputable evidence for the effect of parathyroids upon mouth secretion. This work, likewise, does not seem to have been duplicated for the other endocrine organs. MacCallum and Voegtlin (1908) and numerous later workers have demonstrated the rôle of the parathyroids in calcium metabolism. Marine (1922) and others have adequately shown the interrelationship between all of the glands of internal secretion. While the work of G. E. Smith (1917) and that of Margaret Hoskins (1925) seems to indicate the possible relationship of the thyroid to the teeth, on the other hand thyroid and parathyroids are so intimately connected both anatomically and physiologically as to make it seem likely that the parathyroids could not be completely eliminated, even in such cases.

It is not unlikely that dystrophies of the parathyroids are of primary importance, in the causation of dental diseases, through their influence on calcium metabolism. This effect may be felt both in lowering the calcium content in the teeth themselves, thus predisposing to caries, and in altering the calcium balance of the mouth fluids and the lymph, thus predisposing to the formation of dental calculus and the ensuing soft-tissue lesions. The apparent similar effect of other glands of the endocrine system is very possibly due rather to the effects of dystrophies of these other glands upon the parathyroids, which in turn affect the dental tissues. A considerable amount of careful experimental work is needed to solve this problem.

Summary. This study of 2800 patients served to verify the results of an earlier similar study, which demonstrated that dental anomalies such as missing laterals, peg-shaped laterals, and like conditions were not symptomatic of specific endocrine dystrophies, but were probably genetic in origin. It does, however, indicate the probable rôle of the glands of internal secretion in the etiology of caries and of soft-tissue lesions of the mouth. It is possible that the gland chiefly concerned in this is the parathyroid, through its effect upon calcium metabolism. Other glands possibly bring about the same result through their primary disturbance of parathyroid secretion. Further study upon this problem is intended.

I wish to express my indebtedness to Dr. W. J. Kay and the staff of

the Michigan Home and Training School for their thoroughgoing coöperation, which has made this study possible, and to Dr. George R. Moore for his careful dental studies of the cases presented.

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THE PROBLEM OF CALCIFICATION[1]

Its Influence on the Tenure of Human Life

MAXWELL LAUTERMANN, M.D.

Montreal, Canada

I confess that it has not been an easy matter for me to decide on what phase of calcification I would address you. It is an open secret that this subject is, at the present moment, engaging the attention of a number of physiologists, pathologists and research experts all over the world. And I do not hesitate to state that I have no doubt that many of the problems that have been puzzling us for years will be solved by the way. As you gentlemen are especially interested in treatment of the teeth, it has seemed to me that my remarks, in order to be more practical than polemic, should be along the lines that would be in keeping with your work. You may not have realized the extent to which calcification plays a dominant rôle in the care of the teeth, and especially in the prevention of dental caries.

Calcification presents one of the most interesting problems, in connection with the growth, development, degeneration, and repair of the animal body, with which we are familiar. It plays a prominent rôle in the animal organism from the moment of conception, throughout the period of pregnancy, and after the birth of the individual; from the earliest period of intra-uterine life, when the first signs of ossification occur in the bodies of the vertebrae, until death. It has been considered possible to estimate the age of the individual from an examination of the skeleton, not only of the bones but also of the soft tissues. Especially the arteries show evidence of hardening or calcification, so that the picture of the arterio- and athero-sclerosis is one familiar to every pathologist. Whitacre describes it as a condition where granules of the phosphate and carbonate of calcium are

[1] Read at a meeting of the First District Dental Society of the State of New York, Hotel Pennsylvania, New York City, December 5, 1927.

deposited both in the cells and stroma of tissues. The cause is undoubtedly a preceding degenerative change of the tissues leading to an attraction of lime salts (pathological).

Calcium phosphate, for example, forms three fourths of the total amount of mineral matter in the body, mostly contained in the bones, to which it gives rigidity; it is a necessary constituent of all protoplasm, and is essential to the action of some ferments, such as rennet and fibrin ferments, in the case of the clotting of milk and blood, respectively.

The lime taken into the body with the various food stuffs, under normal conditions, especially flesh, vegetables, yolk of eggs and milk, is sufficient for the needs of the organism. This is what occurs during normal conditions, and accounts for the ossification of bones; it is physiological, but this comment does not take into consideration the pathological calcification. That, as I said before, every pathologist is familiar with as petrification, in which calcification not of new tissue but of tissue already formed and, shall I say, mature, occurs, as in atheroma—a dead or dying tissue in which this process occurs. Here, with the hyaline degeneration and subsequent complete necrosis and liquefaction of the thickened arterial wall, there follows a deposit of calcareous salts, and the formation of calcareous or atheromatous plates in the vessel wall. Similarly it occurs as a secondary degenerative change in tumors, chondromata, fibroids, lipomas, sarcomata. The process may at times be a beneficent one, as in the repair of fractures where the ossein of the newly forming bone is gradually calcified.

Physiologically, calcium salts circulate in the blood in relatively small amounts (about 0.011 to 0.013 per cent of CaO), but in larger quantities than are ordinarily kept in solution. This is accomplished by the colloidal and CO_2 contents of the blood serum, although the colloids alone are sufficient. This greater solubility in colloidal solutions is due to zones of crystalline concentration around each colloid particle. Thus, about two to four times the amounts of tricalcium phosphate and calcium carbonate that are soluble in water are contained in blood. At the same time this balance is easily upset by changes in protein, CO_2, and calcium contents in the blood, but curiously enough, it appears to remain fairly constant in most diseases,

with the exception of nephritis, eclampsia, tetany, and jaundice, when the Ca contents are reduced. Under physiological conditions precipitated calcium occurs only in bone, in the ratio of 85 per cent to 90 per cent of calcium phosphate; a smaller amount, only about 10 per cent to 15 per cent of calcium carbonate; and only a very slight amount (about 1.5 per cent) of magnesium phosphate. This constancy in ratio of calcium salts is reproduced in pathological calcifications, where Ca is laid down in tissues which normally do not contain solid Ca.

Two types of pathological calcification may be recognized. One is a general one, due to increase in the Ca of the blood. Here occur multiple foci of calcium precipitation in parenchymatous organs, principally stomach, kidneys, lungs. This calcification occurs, therefore, in diseases in which calcium is resorbed from normal Ca depots, that is from bones, as in osteomalacia. In osteomalacia, bones soften and discharge their calcium salts (halisteresis). Calcification follows in organs which secrete acid, and in which the tissue fluids are of high alkalinity. Thus, calcification is found under these conditions in the lung, where carbonic acid is given off; in the stomach, where hydrochloric acid is secreted; and in the kidney, where acid salts are eliminated (metastatic calcification of Virchow). But local calcification of tissues is quite different and has nothing to do with calcium contents of the blood, or with reactions of body fluids. This type of calcification is dictated entirely by local environment, and here the circumstances are almost identical with physiological calcification of bones. In each there is a homogeneous matrix with feeble circulation. Thus, degenerated, necrotic, hyaline tissues furnish the ground scar tissues (encapsulated abscesses or tuberculous foci). And such calcified, pathological areas may then show even bone formation, just as normal cartilage, in endochondral ossification, becomes osteoid after calcification. In both instances, a vascular granulation tissue erodes the calcified parts—cells of granulation tissue become osteoblasts and develop bone. Even bone marrow may then arise from connective tissue. This bone-forming influence is not due to Ca, as it does not occur from introduction of Ca free from bone flaps (Gideon Wells).

Now the question arises: what is the mechanism of calcification? It was thought at one time that the phosphoric acid of the blood

combined with calcium, after primary formation of fatty Ca soaps, by replacement with the stronger, inorganic-acid radical. This view is no longer entertained, since neither Ca soaps nor any transformation of such soaps into salts have ever been satisfactorily demonstrated. The same doubt exists as regards the formation of insoluble calcium albuminate. The chemical explanation, therefore, is not well founded. It seems similar to what occurs in incrustation (Gideon Wells), but should not be confounded with crystallization, where it is necessary to have saturation of salts. It appears thus to simulate or resemble adsorption, by colloids with smooth surfaces (for example, gelatin discs), of salts in alkaline media and in low CO_2 content. Cartilage possesses, in similar manner, adsorption affinity for urates of NaCl. It would seem, therefore, that calcification belongs to the group of surface phenomena. It depends primarily upon concentration of Ca in solution on specific smooth colloidal surfaces. This concentration leads to saturation and precipitation of the salt on this surface. While calcified tissues assume stone-hardness after death, it appears from the investigations of MacCordick that Ca salts exist in tissues during life as soft masses like unset mortar. Hardening occurs only when this mass is acidified, as by CO_2, after death.

CURE OF TUBERCULOSIS OF THE LUNGS BY INDUCING CALCIFICATION

As early as 1877, Dr. Both of New York stated that this method of curing tuberculosis of the lungs was suggested by the occurrence of such cases in nature, and he proceeded to ascertain whether calcification could not be induced artificially. He commenced treatment by clearing the bronchioles and vesicles by suction. The result of this is a long, deep cough, with increased expectoration, which is at first of a slimy mucous character, afterwards more dense and cheesy—at times bloody. The muscles are exercised in order, according to their layers and their proper actions. Neither machines, clubs, nor dumbbells are employed, the weight of the body alone being used to increase the power of suction, which becomes greatly increased by practice and correct application. In the case of consumptive patients, with nearly paralyzed muscles, their use requires a great deal of careful and intelli-

gent guidance on the part of the physician: and attention must be paid to the mental, physical, and nervous condition of the patient. Too much force causes pleurisy and hemorrhage, too little is without effect on the extreme parts of the lungs. This constitutes the local treatment. The constitutional treatment is more difficult and complicated. The state of the chylopoietic viscera must be attended to, the products of decomposition in the blood oxidized by the use of citric or other vegetable acids, and lime and silica freely supplied to the organism by the consumption of vegetables containing them. He attributes the value of the old remedies, extract of graminis and album graecum or the white excrement of dogs, to the organic salts of lime they contain. Another lime containing plant is the Achillaea millifolia. Thistles contain silica, and plants growing in swamps contain sulphates. The results of eighteen years' experience led him to the conclusion that no case of chronic affection of the lung is curable, unless vesicular respiration can be re-established: and that no case is curable in which a nidus of pus exists outside the lung tissue proper, whether this be a lymphatic gland or abscess in some other part of the body. The average time required for a lung to calcify is about six to twelve months. (*New York Medical Record*, Feb. 17, 1877.)

"Metabolism" is a big word and covers a multitude of sins. We at last realize that we are physico-bio-chemical laboratories, and unless one understands something of the chemistry of life, it is manifestly impossible to be familiar with the body in health or disease. The calcium metabolism is governed and controlled by the endocrine system: "Man is what he is by virtue of his endocrine glands;" the system of ductless glands by their internal secretion affect, excite, and modify the vital processes of the whole organism.

Parathyroids, discovered in 1880 by Sandstroem, sometimes called Sandstroem's glands, are four in number: the two superior are in close relation with the posterior lateral walls of the thyroid gland, and are sometimes called the internal parathyroids; the inferior parathyroids are situated on the posterior surface of the lower portion of the thyroid lobes, and are termed the external parathyroids. Supernumerary lobes are occasionally found. They are ductless glands, and the method of entrance of their secretion into the systemic circulation is unknown. Gley in 1891 and Loeb in 1900 studied these glands, but to

McCallum and Voegtlin belongs the credit of having established the theory of the parathyroid control of calcium metabolism and tetany. The suprarenals, bone marrow, thymus, spleen, liver and ovaries all take part in activating these glands. Bandler says the rôle of the parathyroids must be of importance in pregnancy, as the ovary and thyroid are activated. It is therefore easy to see how the development and growth of the foetus, and especially the bony growth are, so to speak, supervised by this set of glands. "A child a tooth" is an old and more or less trite expression. If the mother during her pregnancy does not get sufficient calcium in her food, the teeth have to contribute their quota, with the result that their own condition becomes very much impaired.

Vitamins play a dominant rôle in diet and nutrition and are, at the present moment, being investigated by an army of scientists and research workers, the only kind of an army that ever does any good to my way of thinking. It is now well recognized that substances or properties are contained in some foods, especially in leguminous plants, in grain, rice and in fruits (apples, tomatoes, orange juice) which—while themselves not food stuffs—are nevertheless essential for the growth and proper nutritive economy of the animal body. These substances, which cannot be prepared by the body but must be supplied from outside, are termined vitamins. These must be present in a certain minimum amount—just like iodine, calcium, magnesium and iron. They may be extracted and separated by their solubility, some in fat (vitamin A) and some in water (vitamin B). Their absence or withdrawal is responsible for a whole group of so-called deficiency diseases, such as lack of growth, xerophthalmia, scurvy, beri beri, rickets, pellagra. Of the vitamins A is anti-ophthalmic; B anti-scorbutic; D, anti-rachitic; E affects reproduction.

Graham Lusk, in his work, "The Science of Nutrition," published in 1917, said: "In another decade the development of scientific knowledge will probably permit the formulation of this subject from the standpoint of physical chemistry. It cannot now be so treated." Lusk was a prophet. And while we have not developed to the degree that he has outlined, we are nevertheless on our way there; and recent work done in different parts of the world on rickets, scurvy, beri beri and pellagra indicate what we may hope to attain through the science of dietetics, when it is properly understood.

Let me briefly refer to the work done by Deeks, now of New York but formerly of the Canal Zone and Montreal, on this subject, as well as on the inorganic salts in diet. His monographs, "Diet and disease" and "The influence of nutrition on deformities and decay of the teeth," are classics with which every person who presumes to practise either medicine or dentistry should be familiar. Percy R. Howe, of Harvard, with the assistance of Dr. Wolbach also late of Montreal, has been doing some work which is being published in the *Dental Digest*. This scientist points out with great emphasis the important rôle that vitamin C, water soluble, plays in the development of scurvy—not the scurvy that we read about as affecting sailors deprived of fresh vegetable food at sea, but that milder form which many of our wealthy patients suffer from when they come to us with spongy, receding gums, lowered vitality, impaired circulation, leading to the deposit of calcium on their teeth. These symptoms are at first microscopic—the period at which they should be diagnosed and treated.

Let me read you abstracts of two papers by Fujimaki that were reported in Transactions of the Sixth Congress of the Far East Association of Tropical Medicine, published in Tokyo in 1925.

"Formation of urinary and bile duct calculi in animals fed on experimental rations. Report 1, with a short note on treatment, by Yoshitomo Fujimaki. From the Imperial Government Institute for Nutrition, Tokyo, Dr. Tadasu Saiki, Director.

"During the early years of investigation on the vitamin content of Japanese food materials, I recognized a number of urinary and biliary calculi among rats kept a long period on a vitamin A-poor or -free diet. I thought that there must be a certain relationship between the formation of the concretions and diet, especially vitamin A and minerals, and in pursuance of this idea I carried out the following experiments.

"*Experiment 1.* (a) 107 albino rats were fed on a vitamin A-deficiency diet from 2 to 376 days and observed for the formation of concretions; 41 out of 107 rats were kept on that diet for a period of 2 to 30 days, but no calculi were found; 13 rats were kept on that diet for 41 to 50 days, and there were found 3 cases (23%) with bladder stones, but none with kidney or bile-duct stones; 16 rats were similarly treated for 51 to 80 days, and 6 cases (37%) of bladder stones and one case (6%) of kidney stones were found, but no bile-duct stones; 6 rats were kept on that diet for 81 to 100 days, and 5 cases (83%) of bladder stones and 2 cases (33%) of kidney

stones were found, but no bile-duct stones; 8 rats were kept on that diet for 120 to 140 days, and 8 cases (100%) of bladder stones, 6 cases (75%) of kidney stones, and 1 case (12%) of bile duct stones were found; 8 rats were fed for 150 to 210 days, and gave 7 cases (85%) of bladder stones, 6 cases (75%) of kidney stones, and 5 cases (63%) of bile-duct stones; 6 rats were fed for 245 to 376 days, and all had bladder stones (100%), 5 having (83%) both kidney and bile stones.

"(b) 30 so-called Chinese mice were fed on the vitamin A-deficiency diet for 2 to 295 days; 21 out of 30 mice were dead within 31 days, none having calculi; 4 mice were kept on the diet 58 to 65 days, and 2 cases (50%) of bladder stones were found, but no other stones; 3 mice were kept on the diet for 112 to 121 days, and all had bladder stones (100%), one had kidney stones (33%), but there were no bile-duct stones; one mouse lived 295 days and it was found to have bladder, kidney, and bile-duct stones.

"(c) 5 puppies of the same litter and one grown dog were fed on a vitamin A-deficiency diet for 9 to 101 days; 5 of them were either dead or were killed within 71 days, but none had any calculi. One puppy lived for 101 days and was found to have both bladder and kidney stones.

"*Experiment 2.* 178 albino rats were fed on vitamin A- and C-deficiency diet for 3 to 238 days; 79 out of the 178 rats were dead within 30 days, but without any calculi; 16 rats were kept 31 to 40 days, and with 2 cases (12%) of bladder stones only; in 30 rats kept for 41 to 50 days, 5 cases (17%) with bladder stones were found, but no other stones; 27 rats were kept for 51 to 80 days, producing 9 cases (26%) with bladder stones and 2 cases (7%) with kidney stones, but no bile-duct stones; 8 rats were fed for 101 to 122 days, producing 6 cases (75%) of bladder stones, 4 cases (50%) of kidney stones and 2 cases (25%) of bile-duct stones; 4 rats were kept for 141 to 200 days, with all (100%) having bladder stones, and 3 (75%) having both kidney and bile-duct stones; 5 rats were kept for over 207 days with all (100%) having bladder stones and 4 having both kidney and bile-duct stones.

"*Experiment 3.* 38 albino rats were fed on a vitamin B-deficiency diet for 9 to 279 days, but in none were any calculi found.

"*Experiment 4.* 56 albino rats were fed on vitamin C-deficiency diet for 21 to 283 days, but no calculi were found except one case. This rat showed symptoms of xerophthalmia on the ninety-second day, but in two weeks it became well and lived for 159 days.

"*Experiment 5.* 27 albino rats were kept on the vitamin B- and C-deficiency diet for 4 to 169 days showing no calculi.

"*Experiment 6.* 26 albino rats were kept on a normal diet for 56 to 356 days, but did not show any calculi.

"*Chemical composition of the stones.* Repeated chemical examinations of the urinary stones found among the animals fed on the vitamin A-, and vitamin A- and C-deficiency, diets showed the stones to consist mainly of bone earth (phosphate of Ca and Mg), triple phosphate, and a trace of Na, K. The bile-duct stones consisted mainly of cholesterol and pigment.

"*Treatment of experimental calculi.* The rats that were fed on the vitamin A-deficiency diet, and on the vitamin A- and C-deficiency diet, were examined for formation of calculi by x-ray and there were selected 6 rats, in which large bladder stones were found. The diet was changed to a vitamin A-rich diet consisting of dextrin, 65%; casein, 18%; olive oil, 7%; cod-liver oil, 3%; salt mixture, 5%; dried bottom yeast, 2%; daikon juice ad libitum. Besides this diet, about 2 grams of dried river lamprey (Entosphernis japanicus) was given per day, although the animals ate only 1 to 1.5 grams a day. (According to our investigation, dried river lamprey is rich in vitamin A, 0.1 gram per day being sufficient to cure xerophthalmia in albino rats.) After supplying the above diet, the physiological conditions greatly improved and became almost normal.

"One rat was kept on a vitamin A-rich diet for 2 months and then killed and the calculi examined; 4 of the bladder calculi had disappeared, but a small one was left. But this stone's edges were smooth and left a number of smooth angles showing it was a disappearing form; 3 rats were kept on the same diet for 3 months and killed, but no calculi were found, except that in one case soft cartilage-like substances were discovered; 2 rats were kept for 4 months and killed, but no stones were found.

"Formation of urinary and bile-duct calculi in animals fed on experimental rations. Report 2, by Yoshito Fujimaki. From the Imperial Government Institute for Nutrition, Tokyo, Dr. Tadasu Saiki, Director.

"In the first report, I have shown that concretions are found among animals which are kept on a vitamin A-, or a vitamin A- and C-deficiency, diet. In the following experiments, I endeavored to find the influence of protein, inorganic phosphorus, and calcium on the formation of the stones.

"*Experiment 1.* 20 albino rats were kept on a vitamin-A-and-protein-deficiency diet for 3 to 103 days; 14 rats died within a month and 2 cases (14%) of bladder stones were found, but no other stones; 6 rats lived for 31 to 103 days, with 2 cases (33%) of bladder stones and 1 case (16%) of gall-stones.

"*Experiment 2.* 20 albino rats were kept on a protein-deficiency diet (plus adequate vitamin A) for 3 to 101 days, but no calculi were found.

"*Experiment 3.* 25 albino rats were kept on a vitamin-A-and-inorganic

calcium-and phosphorus-deficiency diet for 7 to 64 days; 17 out of 25 rats were dead within 30 days, and among these 1 case (6%) of bladder and kidney stones, and 2 cases (12%) of bile-duct stones were found; the other 8 rats lived for 31 to 64 days—6 cases (75%) of bladder and bile-duct stones, and 4 cases (50%) of kidney stones, were found.

"Urinary stones found among the animals fed on a vitamin-A-and-inorganic-P-and-Ca-deficiency diet consisted mainly of carbonate; and the bile-duct stones consisted of calcium salt, besides cholesterol and pigment.

"*Summary:* (1) Animals kept on a vitamin B-deficiency diet, vitamin C-deficiency diet, vitamin B- and C-deficiency diet, protein-deficiency diet, and on normal diet, did not show any calculi.

"(2) Animals fed on a vitamin A-deficiency diet, and on vitamin-A and C-deficiency diet, for a long period showed calculi, although bladder stones formed in a relatively short period, then kidney stones, bile-duct stones taking the longest time.

"(3) Animals kept on a protein-and-vitamin A-deficiency diet formed calculi in a little shorter time than those mentioned above (2).

"(4) Animals kept on a vitamin-A-and-inorganic-P-and-Ca-deficiency diet formed calculi in a short time. In this case, bile-duct stones formed first, and then bladder stones, kidney stones taking the longest period of all.

"(5) Urinary calculi of (2) were phosphatic calculi, while the calculi of (4) were carbonate calculi; and bile-duct stones of (2) were cholesterol stones, while the stones of (4) were Ca, cholesterol, and pigment."

Dr. Robert L. Porter, of the University of California, finds, after a visit to the medical schools of Italy, France, Great Britain, Canada, and the United States, that everywhere the point of view of physicians is changing to the idea that they should strive to prevent sickness through periodic examination, school hygiene, programs, and general hygienic training. I have taught this for 32 years.

In China it appears that the physicians are paid to keep the people well and that with the onset of any illness their source of income is cancelled. The Metropolitan Life Insurance Company, one of the largest of the life insurance companies, realizing that it was to their advantage to keep people alive as long as possible, established a very active and aggressive health department, and are reaping the results in increased dividends through the premiums that their subscribers, who under ordinary conditions would have died at a much earlier age, are

only too glad to pay. It should be our ambition to be health engineers and not just repair men. I believe that you of the dental profession realize that in treating the teeth you are only dealing with one of the tissues of the body, and that the disability which attracts your attention to that part is only an index of what is going on in other parts of the body and should be so considered.

One of the most prominent factors in maintaining health and efficiency is what we speak of as a balanced diet. We should bear in mind that the acid and alkaline balance as well as adequate vitamins, amino acids, alkaline inorganic salts and water are the elements which build tissues. Vitamines, especially A and C, and inorganic salts in cereal foods, which are removed in the process of milling, are essential to health. The hull and outer layer, which constitutes the bran, removes the vitamins and lime salts, leaving the devitalized and devitaminized starch or carbohydrate behind to be made into white bread. The correction of this deficiency is not an easy matter, but it can be done and I believe is very much worth while. I believe that peace has its victories even greater than those that may be won at war.

You should realize that the lives and welfare of the future generation are in your keeping, and that the care given the child today is represented by the harvest that the man of tomorrow reaps. The care of the unborn child and its mother (for she is the crucible of life) are in your hands. The extraction of a diseased tooth or the removal of a diseased appendix does not represent the highest aim of our art. When I tell you that appendicitis can be avoided, that kidney and gall stones can be removed without operation and their recurrence prevented, I am not dreaming. Pay a lot of attention to men like Deeks, Howe, your own President Chayes, and McCollum, who are pioneers each in his own sphere, to whom we owe a greater debt of gratitude than we can ever hope to discharge.

Hear what Dr. Victor Heiser, director for the East International Health Board has to say in the "*Military Surgeon*" for February last:

"The Japanese have made enormous progress in the field of nutrition; and we can look to them for great contributions to modern medical knowledge. While making a survey of the entire health service for the Japanese Government, I saw among other things the

work of the Imperial Nutrition Laboratory. I hesitate to tell you of perfectly astounding things I saw in the nutrition laboratories. Some of them have not yet appeared in the medical journals. The Japanese produce stones in the gall bladder, kidney or bladder, by diet, and dissolve them by a reversal of the diet. In the majority of cases they could fill stones in the particular organ they desired and dissolve them again. They also produce gastric ulcers with great precision and could turn them into cancer. You can see what great possibilities these things hold for research in the future. I do not claim to be a pathologist but I did look at specimens under the microscope which to me appeared to be typical cancer. Men present at an International Medical Meeting, who were skilled in Pathology, pronounced the specimens cancer.

"Most experiments have been done on white rats. I am more and more impressed with the fact that there may be something very important in connection with nutrition in the control of tuberculosis. I wonder whether the measures which have been invoked for the control of tuberculosis are the very things which produce the results. Rats are resistant to tuberculosis, and guinea pigs are very susceptible. Feed rats a vitamin A-deficient diet and they become as susceptible to tuberculosis as guinea pigs. That may or may not have significance in connection with human tuberculosis."

"In India I saw huge dilated stomachs in animals produced by a definite diet. In India there are people who grow to great physical proportions, living among others and apparently of the same economic condition. Colonel MacCarrison of the Indian Army studied this matter, and found that the diet of these large people is almost ideal, containing the requisite amount of inorganic salts and vitamins. Experiments have been made with rats by feeding them the characteristic diet of the large people, and English diet, French diet, and Italian diet. It is astounding to look at these rats. The rats fed on the Sikh diet of the large people of India grow to huge proportions with silky hair, while the English-workman rat is scrawny, of fairly good size with a rough coat."

Some time ago, we conducted a little experiment at an orphan asylum. We fed all the children the same diet with essential vitamins and inorganic salts. At the end of a year, we had only half as much

tooth trouble, cough, cold, and indisposition. Those in charge said it was ungodly and that they could not manage the children any more because they became too wild. I wonder sometimes when I go through an asylum whether the children are well behaved because of discipline or diet.

I have been impressed with the whole question of the possibility of food being a vital factor in the prevention of tuberculosis. I do not say that it has a real relation. I do venture to say that if you show a curve of improvement in tuberculosis you will probably find another curve showing that milk consumption also has increased. The milk consumption in the United States is almost a pint per capita. That means practically one hundred ten million pounds of milk used in the United States every day. People with such a wholesome food in such large quantities are bound to improve in health. The people of Holland and Denmark have a lower death rate than we have, and there you find them living largely on milk and milk products. Moreover, they do not have the health organizations which we believe necessary in this country. Let me emphasize that milk in its natural and pure state is the most perfect of foods. Rich in calcium, a quart of milk contains more Ca than a quart of lime water; it is rich in vitamins A, B, and C. This latter vitamin is absent in milk in winter because cows do not get fresh green food. This vitamin is found in fruit and vegetables grown in the open air, and is so delicate that fruit and vegetables grown under glass contain a very much smaller quantity.

Coöperate with the investigators of today to help the public which is so much in need of honest, intelligent help. Realize that instead of the body having resistance to disease, the cell has permeability to the influence of osmotic energy; that high blood pressure, for example, is a symptom of disease but not in itself a disease; and moreover that it can be treated with satisfactory results to the patient and the physician as well.

DISCUSSION

William E. Deeks, M.D. (New York City): I am not going to discuss Dr. Lautermann's paper, but I shall add to or supplement it with one or two remarks which may be of use to you in your practice. There is no question that there are two or three main stumbling blocks in the regular diet at

present in general use, and it is those stumbling blocks to which I want to draw your attention.

In the first place, we must consider the inorganic salts in our diet. We have the acid-ash foods, and we have the alkaline-ash foods. These points that I am going to refer to are not generally known among physicians. I belong to a profession allied to yours, and I do not think that our contacts are sufficiently great. You are merely taking care of one of the tissues of the body, other specialists in the allied profession are taking care of other tissues. And I do not believe that those who are the diagnosticians or the internists in the medical profession are sufficiently alive to the situation because even most of these are beginning to specialize. Thus we are getting away from the old-fashioned diagnostician, our family physician, if you will, who directed his patients to those specialists who were best able to take care of them.

Going back to inorganic salts: the acid-ash foods are the flesh foods, whether they be meat, fish, or poultry. In addition to this, tea and coffee, and cereals of all kinds and everything that is derived from them, are acid foods. In other words, cereals are acid-ash foods. Eggs are also acid-ash foods, much more so than meat. Oysters have about the highest acid-ash that we know.

Let us now take the other group. Where do we get our alkali? Our blood is alkaline, and we must preserve the alkalinity if we are going to preserve our health. The alkaline foods are the root vegetables—anything that grows under ground. The second great group is the legumes—peas, beans and lentils, whether dried or green. Of all these lima beans are the most alkaline food we know. The other foods are the leafy green vegetables and the fresh fruits. It is in the vegetables, leafy green vegetables, and fruits, particularly, that we get our supply of vitamins, and here is where we are making the mistake today. The people of this country are eating too much of the cereal foods. I condemn nearly all cereals except for children—and that has got to be controlled, that is the breakfast cereals.

You will not only find gallstones, kidney stones, appendicitis, and constipation, but a great deal of the dental decay, in people who are eating excessive amounts of breads, which are acid-ash food. That is particularly true since the rolling-mill process has come to the fore. Now instead of grinding our wheat between stones, we are bolting it—removing absolutely all the vitamins, and practically all the inorganic salts. I do not condemn white bread, but if you are eating a lot of white bread, you must eat a lot of vegetables and fruits, or the germs of disease will early take hold of you.

Your teeth are merely modified epithelium. So is your hair. I am

going to advise you who are prematurely bald—and I am a victim, too—to look into your diet. I am going to advise you who are having periodical headaches to look into your diet. You may get Bright's disease under the conditions that favor development of the stones to which Dr. Lautermann referred. You must look into your diet if you are going to prevent early decay of the teeth. The unfortunate part of it is that when your teeth begin to decay—and remember that teeth are a tissue—the same processes are beginning in other tissues, but in an insidious manner, and some day you will wake up to find arteriosclerosis, kidney stones, and a variety of things of that sort.

Take the people, between fifty and sixty years of age; who are dropping dead here in the street today. They are merely cases of myocardia degeneration going on over a period of years through deficiency diet. If these deficiencies are corrected early enough you can save the patient. It is an example of what is going on all over the body. It is the same thing with high blood pressure. Don't eat more than four slices of bread in the day as a maximum. Take the rest of your starchy foods in root vegetables, potato, and other green vegetables. You may eat meats once or twice a day. It is in the starchy group of foods, particularly, that we find the foundation today, the etiological factor, for early tooth decay as well as the degeneration of other tissues.

MEDICO-DENTAL CASE RECORDS: SIXTH REPORT[1,2]

A Clinico-Pathological Study

BISSELL B. PALMER, Jr., D.D.S., F.A.C.D., and MALCOLM W. CARR, D.D.S.
New York City

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I. Introduction

The medico-dental case records in this series of reports have been limited entirely to the sequelae of oral *focal infection*. This report of a case of cancrum oris, which is a severe local destructive process by direct extension, does not, therefore, come strictly within the set limitations of the present serial study. However, because of the many unusual features of this case (principally the incidence of the disease in an adult), and the availability of a complete and detailed chronological clinical record, it is believed that the following report may be of some value to the literature of both dentistry and medicine. Furthermore, this report exemplifies the type of case in which proper treatment can be instituted only by the rational coöperation of the physician and the dentist, for, since the treatment is both local and constitutional, the case falls neither into the practice of medicine alone nor into the practice of dentistry alone.

[1] Presented by title at a session of the Sixth Annual Meeting of the International Association for Dental Research, Hotel Mayflower, Washington, D. C., March 25–28, 1928. An abstract was published in the *Journal of Dental Research*, 1928, viii, p. 449.

[2] The previous reports were published in the *Journal of Dental Research:* (1) 1926, vi, p. 283; (2–4) 1927, vii, pp. 115, 275, 457; (5) 1928, viii, p. 73.

The occurrence of this case antedates the beginning of the present medico-dental case-record study, and in consequence details of general physical examination and laboratory data, which have become standards of this series, are incomplete.

II. Case M-2: Cancrum Oris (Noma)[3]

General history

The patient, a man fifty-five years of age, a building contractor, was seen in consultation on January 23, 1924. His chief complaints were pain and swelling in the jaws, and inability to open them. The condition was caused by an abscess associated with a pericoronal infection of the right mandibular third molar. No data of importance were obtainable regarding early childhood or adult medical history or family history. For several years the patient consumed large quantities of Scotch whiskey daily. For five years he had had gastric reflexes with pain and belching one hour after meals. Three years ago he noticed a double direct inguinal hernia which was corrected surgically soon afterwards. In November, 1923, a medical examination was made and a "hob-nailed" liver was diagnosed. At that time the border of the liver was not palpable below the costal margin, and by percussion it was evident that the lower edge was probably in normal position. There was pain over the liver. There was a slight loose cough, probably due to excessive use of tobacco. The veins of the legs and abdomen were markedly enlarged, probably due to back pressure from an obstructed portal circulation. The lymphatic systems of the neck, axilla, and groin had been enlarged for ten months. Blood pressure readings were: systolic, 125 mm.; diastolic, 80 mm. Heart and lungs were negative. There was no dental history of importance, with the possible exception that, about January 9, 1924, a gold crown which had been irritating the gingiva around the right mandibular first molar was removed and a gold inlay substituted.

Course of disease and treatment

Under nitrous oxide and oxygen anesthesia, chosen because of the acute local condition, the mandibular third molar was extracted and

[3] The condition of cancrum oris is described in detail in the "Supplement," p. 587.

debris removed from the wound, which was then irrigated and dressed with a half-inch width of gauze. There were symptoms of respiratory obstruction during the procedure, owing probably to the inflamed and congested condition of the upper respiratory tract caused by the general stomatitis. The following day the submaxillary and sublingual glands were swollen and indurated; the face also was more swollen, red, and shiny, but without evidence of any skin tension. The temperature was normal and no pus was visible; the wound was cauterized, irrigated, and redressed. On the third day the swelling was less, and general improvement was noted, which was maintained until the seventh day when an exudation of pus was noticed around the right mandibular first molar, from which a gold crown had recently been removed. The original wound was doing well. On the eighth day some evidence of a generalized gingivitis was noticed, a smear was taken, and Vincent's organisms were found. Sodium perborate routine treatment was instituted and the condition improved. The third molar wound was sluggish in healing, probably due to the mixed infection, and was redressed with gauze for the first seventeen days.

On the thirteenth day a roentgenogram of the right mandibular first molar failed to disclose any evidence of a pathological condition. On the seventeenth day the patient was much improved. As he had to make a trip to another city, he was advised to have his mouth treated in that city and to continue the sodium perborate treatment. On the twentieth day the patient returned with increased swelling and pain, and evidence of complete neglect of the mouth, which he admitted. The clinical symptoms of Vincent's infection were now very definite. On the twenty-first and twenty-second days the patient became quite weak and another roentgenogram of the mandibular first molar, made on the latter date, was negative.

On the twenty-third day the patient was confined to bed, and was quite weak. The same routine local treatment was maintained and consultation with the medical adviser of the patient resulted in no change in treatment. There were no developments on the twenty-fourth or twenty-fifth days excepting a tendency of the inflammation to center in the region of the mandibular first molar. On the twenty-sixth day a blood culture was positive for streptococcus viridans. During these days the temperature was only a shade above normal;

but inasmuch as the temperature was being taken by mouth, due to the insistence of the patient because of hemorrhoids, it seems safe to assume that it was probably somewhat higher, as ice was being used on the face to relieve pain. Deep probing around the gingiva of the mandibular first molar disclosed the presence of several thin flakes of dental cement, which apparently were remains of the fastening cement (used at the time the gold crown had been put on) that had subsequently dropped into the pocket around the tooth when the crown was removed. On the twenty-seventh day the teeth were scaled.

FIG. 1. Site of the original ulcer in the alveolar muco-periosteum adjacent to the right mandibular first bicuspid.

On the twenty-eighth day a well marked ulcer, about 0.5 cm. in diameter, developed almost directly opposite the right mandibular first bicuspid (two teeth anterior to the previous center of inflammation—*fig. 1*). The ulcer was about 1 cm. below the gingival margin, was not deep, and did not expose the bone. On the twenty-ninth day further consultation with the physician and another oral surgeon resulted in an effort to combat Vincent's infection with salvarsan locally. The ulcer was deeper, but not wider. On the thirtieth day a slough appeared around the edge of the ulcer. On the thirty-first day the patient felt much better, but the lesion had

extended to a point where it would more suitably be termed a slough than an ulcer. On this day the first bicuspid became quite loose and very sensitive. On the thirty-second and thirty-third days the slough continued to extend; on the thirty-fourth day it seemed no worse, but as all the remaining teeth on the right side had become quite loose and very painful, the remaining right mandibular teeth were removed with local anesthesia. On the thirty-fifth day no change was noted except that pain was less because of the removal of the teeth the previous day. The thirty-sixth day saw the slough again extending; on the thirty-seventh day rapid extension and increased swelling were noted.

FIG. 2. Position on the external surface of the cheek of the typical black spot of noma—the point of perforation.

FIG. 3. Area of destruction of the terminal lesion approximately as it existed at the time of death.

The slough now extended into the reflection of the buccal mucosa, the mandible was exposed in the bicuspid and molar regions; and the periosteum was destroyed at the points of exposure.

On the thirty-eighth day further consultation was held with another oral surgeon. Mercurochrome, 5 per cent, was resorted to in an effort to combat the infection. The remedy was used on gauze and the edges of the sloughing wound were kept comfortably distended with it. By this time rectal temperatures were being taken and had ranged from 100 to 101.4. On this day the temperature suddenly rose to 103.4, and for the next several days ranged between that point

and a degree lower. On the thirty-ninth day the process continued. On the fortieth day one of us made a definite diagnosis of noma, and on the following day advised hospitalization and a wide use of the actual cautery. In consultation, it was deemed inadvisable to resort to this procedure, because of the poor general condition of the patient. It was decided to attempt to control the condition by using mercurochrome, 1 per cent, intravenously. This remedy had recently been enthusiastically offered to the profession, and it was thought that some benefit might come from its use. The temperature was 103.4 when 13 cc. were injected. The following day, the forty-second, a seemingly miraculous change had come over the patient. The temperature had dropped to 99.4 for the first time in a number of days, and early in the morning the patient was found sitting up in a chair reading the morning newspaper. The wound looked better, as did the mouth generally. Twelve cubic centimeters more of 1 per cent mercurochrome were injected. There was a marked reaction to this second injection, however. The patient turned suddenly weaker, bordering on collapse, and no further effort was made along this line of therapy.

On the forty-third day there was no evidence whatever of slough, the wound looked clean, and the odor suddenly disappeared. On the morning of the forty-fourth day the condition still seemed to be improving, but in the evening swelling suddenly increased, and the temperature rose a degree and a half, to 102. No perceptible change took place on the forty-fifth day, but a blood culture again was positive for streptococcus viridans. On the forty-sixth day the right eye was slightly swollen and consultation with another surgeon was requested by the family. This consultant disagreed with the diagnosis of noma, believing the condition indicated osteomyelitis. He accordingly advised the use of external heat from radiation lamps. The patient's physician and the writers did not agree with this opinion, but the prominence of the consultant, the confidence which the family had in him, and the obviously desperate condition of the patient made it seem justifiable to resort to a trial of heat, at least for a short period.

On the forty-seventh day there was a perceptible odor and the first evidence of extension of slough in several days. The heat was stopped immediately. At this time the slough had reached the second molar

region posteriorly, and the lateral incisor region anteriorly. It had extended up into the cheek, and had exposed the mandible below the mental foramen. On the forty-eighth day the slough was further extended, and the maxillary molar on the opposite side had set up an independent infection. The wound was very foul, and the odor was noticeable upon entering the sickroom. On the forty-ninth day a dark, almost black, circumscribed area about 1 cm. in diameter appeared on the outside of the right cheek (*fig. 2*). On the fiftieth day a perforation developed in the cheek and was extending rapidly. On the fifty-first day the slough had moved rapidly and a large section of it was removed in an effort to clean the wound, decrease toxic absorption, and facilitate treatment. The extent of slough through the cheek was much greater than the surface symptoms indicated. A further consultation resulted in giving up the mercurochrome treatment locally, and, instead, an electrolytic chlorine solution on gauze was introduced into the area. It was expected to combat the foulness, and hoped to be antiseptic enough to check the bacterial invasion.

From the fifty-second to the fifty-seventh day the patient became much weaker, the destruction continued rapidly, and at times an extension of the slough was noticeable between visits on the same day. On the fifty-third day a culture, taken from a section of slough far up in the cheek away from mouth contamination, was positive for staphylococcus aureus in pure culture. On the fifty-eighth day the slough approached the corner of the mouth. On the fifty-ninth day the destruction came closer to the mouth. On the sixtieth day the process reached the mucous membrane at the corner of the right lip. The patient had been given morphine sulphate at night throughout the illness in gradually increasing doses. The dosage was now increased to 0.5 grain.

On the sixty-first day the slough broke through the right corner of the mouth, causing the destroyed area in the cheek and the mouth to become one continuous cavity, also attacking the edge of the upper lip. On the sixty-second day the slough was extending very rapidly. By the sixty-third day extensive broadening of the area of slough had taken place, including one-half inch of the lower lip and one-quarter inch of the upper lip. On the sixty-fourth day the patient complained of being blind in the right eye. The respirations had reached 28 for

the first time, and the slough had extended one-half inch in all directions overnight. On the sixty-fifth day the slough had gone on for a distance of three-quarters of an inch in all directions and was within three-eighths of an inch of the ala of the right nostril. Slight pulmonary congestion was in evidence. In consultation it was considered advisable to attempt to check the slough by use of a solution of bromine, 1 dram; potassium iodide, 1 dram; and water, 8 ounces. A dram of the solution was diluted in 7 ounces of water and was applied to the gauze dressing in the wound.

On the sixty-sixth and sixty-seventh days the slough had destroyed about one-half of the lower lip, and had extended well across the upper lip. The patient was unable to swallow. The physician advised against rectal feeding as the case was a hopeless one and death was only a question of a short time. On the sixty-eighth day the ala of the right nostril was destroyed (*fig. 3*), the slough had approached the pillars of the fauces, and the sight of the left eye had become affected. The patient had several spasms of the glottis on the sixty-ninth day, indicating the extent to which the slough had extended posteriorly. He was exceedingly weak, although mentally alert, and was receiving one-half a grain of morphine sulphate *ad libitum*; but, getting no nourishment, was very low. After several spasms of the glottis on the seventieth day, the patient expired from acute edema of the glottis.

III. Summary

The incidence of noma in adults is not common, and this history narrates several interesting phases of the disease with regard to possible etiology, point of origin, course, treatment, and termination. The disease process originated from a small ulcer located on the buccal surface of the muco-periosteum adjacent to the right mandibular bicuspid, was complicated by Vincent's infection, spread rapidly through the tissues of the cheek, and resulted in widespread destruction of the face, which extended from the angle to a point approaching the symphysis of the mandible (involving the lower and upper lips almost to the median line, the ala of the nose), and superiorily to the infra-orbital structures and zygomatic process. In the terminal stages of the disease the patient became totally blind in the right eye, and showed severe signs of secondary anemia with marked cachexia.

The primary fatal lesion was the extensive cancrum oris; the secondary or terminal lesion which caused death was edema of the glottis.

IV. Conclusions

1. There was no definite evidence in this case that cancrum oris is caused by a specific organism; however, the temporary improvement after intravenous use of mercurochrome might be construed as evidence that there is a bacteriological factor in the etiology of the disease.

2. No local treatment in this case exerted the slightest retarding influence on the extension of the process of destruction.

3. The use of the actual cautery in this case, in an attempt to circumscribe and arrest the disease, was contraindicated because of the grave operative risks, so that its possible value was not demonstrable; but the recent development of endothermal and diathermal surgery offers the possibility of a procedure which, if used early in cancrum oris, might be justified in the absence of any other definitely proven remedy.

V. Supplement

Definition. Cancrum oris (noma) is an affection characterized by a rapidly spreading and progressive gangrene, usually starting as an ulcer in the alveolar mucoperiosteum or buccal mucosa, which tends to perforate the tissues of the cheek leading to extensive sloughing and facial destruction.

Occurrence. The condition occurs so rarely in those who are past puberty that it is generally considered to be a disease of early childhood, occurring chiefly between the ages of two and eight. The disease occurs almost exclusively in children who are already debilitated by some other disease, most commonly one of the specific infections, especially measles. It may also be observed during the course of or after scarlatina, diphtheria, typhoid, and whooping cough.

It is fortunate that the incidence of the disease is comparatively rare. It is seldom seen in private practice. In institutions it is most frequently observed among poor children who, upon admission, are in a wretched state of health due to malnutrition and unhygienic environmental conditions. Under such conditions children readily fall victims to infections of all kinds. Because of the infectious nature of noma the cases should be isolated when found in hospital wards. It has been observed in epidemic form, in institutions, because of failure to isolate the first case.

Although the disease is most frequently observed in young children and infants it is not unknown in adult life. Brown[4] describes a case of noma in a young man twenty-two years old who always had been in robust health. The point of origin was distal to a mandibular third molar. The disease pursued a rapid course resulting in death in ten days.

When noma is observed in older persons it is almost invariably a terminal infection or an expression of some pathological state of a general character, such as pernicious anemia, leukemia, tuberculosis, or diabetes.

Etiology. Noma is a disease which is undoubtedly infectious in origin. Many bacteriological studies have been conducted in an attempt to isolate a specific causative microörganism, but without success.

The bacillus described by Lingard has been found in a small percentage of cases. Various other pathogenic organisms have been found frequently in the gangrenous tissues of noma, such as streptococci, staphylococci, diphtheroid organisms, diphtheria bacillus, fusiform bacillus, spirillum of Vincent, and a form of streptothrix.

Attention has frequently been directed to the coexistence of Vincent's infection during the course of noma. Appleton[5] states that fusiform bacillus and spirochetes (the fuso-spirillary symbiosis) may appear as the predominant organism in smears taken from the gangrenous sloughs. In this regard Appleton cites the experiments of Tunnicliff in which it was found that the subcutaneous injection of killed fusiform bacilli and spirochetes, in a case of noma, caused an increase in the amount of specific opsonin (elevation of the opsonic index), and appeared to produce some beneficial effect upon the patient. The chart of the opsonic curve illustrating the published report of the experiment indicates clearly a rise in the opsonic index following each such injection. In all probability, however, the gangrene of noma is due not to infection by any single microörganism but to a process dependent upon a mixed infection, in the presence of lowered tissue resistance from general debilitation.

Feebleness of circulation, and interference with it by bacterial embolism and thrombosis, are probably factors that contribute to the progressive spread characteristic of the gangrenous process. There has been some evidence that noma is due to blocking of the circulation of the tissues through invasion by Vincent's fusiform bacillus and spirochete. Hopewell-

[4] Brown, G. V. I.: Surgery of Oral Diseases and Malformations; Lea and Febiger, Philadelphia and New York, 1912.

[5] Appleton, J. L. T., Jr.: Bacterial Infection; Lea and Febiger, Philadelphia and New York, 1925.

Smith[6] states that capillary thrombosis in the cheek, perhaps due to Lingard's bacillus, is probably an important factor.

Morbid anatomy. The disease may supervene upon an ordinary ulcerative stomatitis, or it may develop independently as a small localized ulcer. In the latter event the favorite site is the mucous membrane of the cheek near the corner of the mouth. The early process is an inflammatory reaction with round-cell infiltration and an active hyperemia, but soon, possibly due to a thrombosis of the vessels, gangrene sets in. The cheek is affected first. A dusky red or black spot appears on the buccal mucosa and later on the skin surface of the cheek, which is the site of early perforation, after which the tissues become extensively swollen, edematous, and discolored. The gangrenous process spreads rapidly, both on the external surface, and within the mouth, and sloughing is extensive. There is no apparent effort on the part of the tissues to limit the progress of the gangrenous destruction. The extensive thrombosis of the vessels usually prevents hemorrhage, and, owing to destruction of the nerve filaments, there is comparatively little pain.

Course and symptoms. The process begins insidiously. The earliest lesion may be a simple localized dissolution of continuity in the alveolar muco-periosteum or buccal mucosa that later develops into a small superficial ulcer. The ulcer becomes progressively larger, involving the deeper tissues and periosteum and spreading to the adjacent tissues of the cheek. The side of the face becomes much swollen and distorted, and the tissues when felt with the fingers are brawny and hard. The submaxillary and cervical lymph glands become extensively involved, being swollen and indurated. Salivation is marked. Frank pus is seldom seen. The black gangrenous masses form so rapidly that although the wound be cleansed four to six times a day, the dead tissue may be wiped off with cotton swabs at each attempt. It is useless to excise the dead tissue, because the gangrenous process begins immediately at the border of incision.

The teeth are loosened and exfoliated, the periosteum is destroyed, and the maxillary bones become necrotic. An unbearably foul and nauseating odor emanates from the gangrenous area due to putrefaction of the tissues. Although there is great tenderness and pain from pericementitis and periostitis during the early stages of the disease, there is little discomfort from pain during the terminal stages.

Thus a large portion of the side of the face may be destroyed. The gangrenous process may extend into the orbit, the nose may be obliterated,

[6] Hopewell-Smith, Arthur: Normal and Pathological Histology of the Mouth, Vol. II; P. Blakiston's Son and Co., Philadelphia, 1918.

and the normal orifice of the mouth becomes continuous with the cavitation of the cheek.

The general symptoms are severe, requiring stimulating and sustaining treatment. The appetite is lost. The pulse becomes weak and thready, and the rate accelerated. The temperature, although often high at first, (102°–104°) is usually subnormal before death. The respirations become rapid and shallow during the terminal stages. The patient appears markedly septic and cachectic. There may be profound depression and exhaustion, although mental alertness may be manifested until a few hours before death.

Prognosis. The prognosis of cancrum oris is exceedingly grave. It has been estimated that 80 per cent of the cases terminate fatally in from ten days to two weeks. When recovery occurs it is usually with marked deformity from cicatrization.

Noma results in death because it exists as a primary fatal lesion as well as a secondary or terminal lesion, that is, one which develops only in those whose vital resistance is so lowered by constitutional disease or poor nutrition that the pathological process is possible.

Bronchitis and terminal (hypostatic) pneumonia are likely to exist during the last stages of the disease; and when death supervenes, the patient is usually in a state of utter exhaustion, due to toxemia and septicemia.

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667 Madison Avenue.

A NEW CABINET—BRACKET TABLE—TRAY METHOD IN DENTISTRY[1]

WILBER M. DAILEY, D.D.S.

New York City

In the average dentist's office, dentistry is not scientifically practised. I therefore present for your consideration a new method. This new method is as follows:

(1) A new design of cabinet for holding thirty-two enameled metal trays; a specially adapted apparatus for their sterilization.

(2) A new design of bracket table.

(3) A new method of procedure in the practice of dentistry through the use of these new scientific devices.

This cabinet—bracket table—tray method is scientific and practical, decreasing the time consumed in all classes of operations—a protection for patient and dentist, making aseptic surgical dentistry available for every practitioner.

(1) The *new cabinet* is five feet, one inch high, and three and one-half feet long. It is made in two parts to facilitate moving. The upper part is fourteen and one-half inches deep and the lower twenty-four inches. It is entirely of enameled metal and glass.

The lower section has a series of drawers (two deep) extending across the top, and a series on each side with space between for a chair. The top of this lower section is a heavy, white-glass slab, serving as a base for the upper section. Under this slab are three sliding, enameled-metal shelves to hold trays or basins for surgical operations.

The upper section of the cabinet is composed of three compartments. The two side compartments are especially designed for holding enameled-metal trays, sixteen on each side. The doors are of metal and glass, and are made air tight by a padding of special felt. Under

[1] Awarded the Lord Prize for 1927 by the First District Dental Society of the State of New York.

the bottom tray in each compartment is a small chamber holding a formalin lamp, the space between the side of the cabinet and the narrow tray-supports permitting the circulation of the formaldehyde gas. There is also a special funnel and pipe with a cut off, to allow liquid ammonia to be poured from outside the lamp chamber into a dish beside the lamp, thus neutralizing the formaldehyde gas fumes before the doors to these sterile tray compartments are opened, formaldehyde gas being intensely irritating to the eyes and air passages.

The middle compartment is fitted with glass shelves to hold the various supplies used in special operations. This also has metal and glass doors, and doors on all compartments have special locking devices.

The accompanying photographs (*figs. 1, 2, 3* and *4*), showing these compartments, indicate the efficient service rendered by this new departure in cabinets. The cabinet may be made with one formaldehyde chamber, or in any size desired.

(2) The *new bracket table* is an oblong piece of quarter-inch glass framed with a detachable, nickel-plated metal rim, which holds in place the paper cover for the table. On each side of this rim runs a groove into which is fitted a sliding holder for an enameled metal tray. This holder is five inches high and an entirely new feature. The table accommodates two trays, and the third tray, being elevated and movable, not only enlarges the capacity of the table, but permits the unobstructed use of the two trays upon it.

Photographs (*figs. 5, 6, 7* and *8*) show the table without the upper tray and with it; and the table rim with groove and the holder for the upper tray. Note the wheels set into the base of the holder, on which it moves and which prevent any retardation of motion.

(3) *Method of procedure.* Knowing that dental operations can be performed in an aseptic surgical manner, I devised the cabinet and tray method, having sterilized compartments to facilitate constant readiness, for immediate use, of a series of sterile, duplicate trays holding duplicate sterile sets of instruments and accessories for the progressive steps of each common operation, e.g.: a number of trays having dental napkins, a special dish (blue) for absorbent paper pellets, a special dish (white) for waste, sheets of paper for use in

FIG. 1. The new cabinet

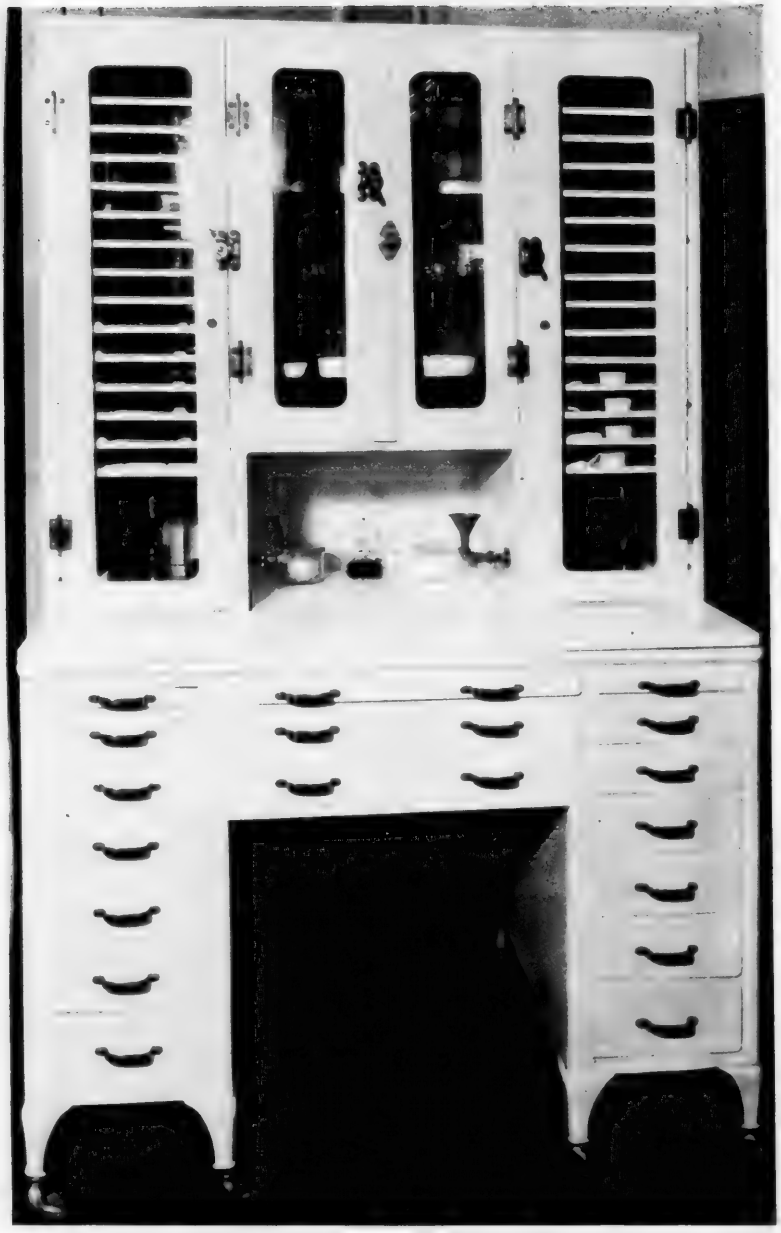

FIG. 2. The new cabinet; a second view

FIG. 3. The new cabinet, showing compartments, trays in formaldehyde chambers ready for use, formalin lamp, and ammonia neutralizer

FIG. 4. The new cabinet, showing sliding drawers to hold basins for surgical operations. The two open drawers show how metal containers can be used for small supplies, inlays, etc.

changing hand pieces (so the chain of asepsis will not be broken), cotton rolls, cottonoid pads.

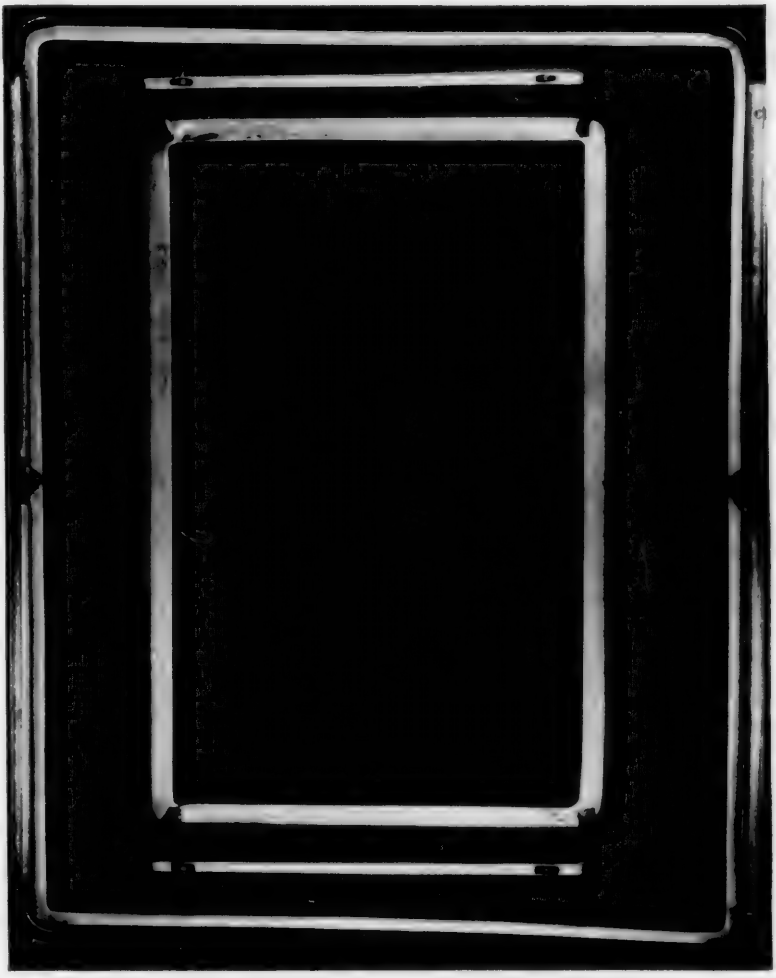

FIG. 5. The rim of the new bracket table showing space for the upper section base to run in, the upper section showing a support with rollers to permit sliding

A number of other trays may be made as follows:

Hand pieces, straight and right angle; a mouth mirror; a pair of

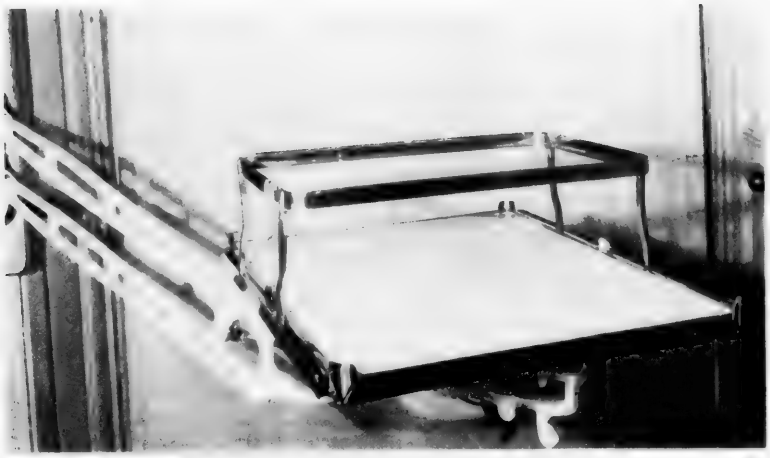

FIG. 6. The new bracket table in position

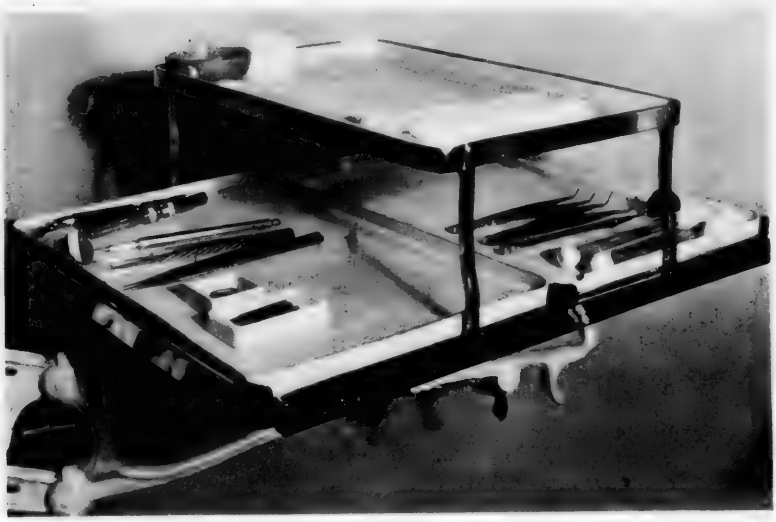

FIG. 7. The new bracket table ready for use

pliers; two examining instruments (Oo-o Grafrath's); a right angle excavator and a 45 degree excavator.

A number of trays for special sets of amalgam instruments.

A number of trays with instruments for pulpless teeth operations.

FIG. 8. The new bracket table ready for use; a second view

A number of trays with instruments for special operations, e.g.: gold fillings, gold inlays, etc.

These sets can be made up to suit the pleasure of the individual operator.

The trays, instruments, and contents are sterilized before being placed in the cabinet, but are re-sterilized after placing them in the formaldehyde chamber to afford a double check.

The purpose of the special bracket table and cabinet method is to afford the operator and patient ample aseptic surgical protection. Also to facilitate a saving of time for the dentist in his operations by having all his methods standardized, so that, by the prearrangement of trays to meet his needs as he takes each step in his operation, he can proceed in an orderly and systematic manner.

NON-OCCLUSION AS A FACTOR IN PERIODONTAL DISEASE

SAMUEL CHARLES MILLER, D.D.S.

New York City

Many pages have been written on the importance of traumatic occlusion as a prime factor in the production of periodontal disease. The presence of this disturbing factor is a common one and may be said to be outstanding in at least 85 per cent of the cases which are brought to the attention of the periodontist. The treatment of these cases has been thoroughly investigated by our ablest periodontists and the outline of the technique for the elimination of traumatic occlusion has been simplified so greatly that, with the technical skill of the average practising dentist, most satisfactory results may be obtained in the eradication of this disturbing factor. There is, however, a group of cases the occurrence of which is much less frequent, but which is seen often enough to cause trouble unless we can avail ourselves of some treatment that will both clean up the periodontal disturbance and prevent its recurrence, as well as establish a functional relation of the teeth that were so affected.

Non-occlusion is a condition in which one or more teeth cannot be brought into contact with their opponents in any position that the mandible may assume. The non-occlusion may be either congenital or acquired, and may occur between single teeth, or two, three, four, or more teeth. A condition which Dr. John Bethune Stein has termed "artichoke bite" consists of the entire absence of occlusion on the anterior teeth, and is often seen in conjunction with hypoplasia of the anterior teeth (Hutchinson's teeth; *fig. 1*). This condition may be noticed in teeth of apparently normal form (*fig. 2*), and is apparently due to an arrest or slowing up of the growth of the anterior alveolar bone. In some instances a more rapid eruption of the posterior teeth may be responsible. Cases are fairly common in which the anterior teeth are malarranged, either the upper teeth being in labioversion, the lower teeth being too far to the lingual, or a combination

of the two conditions may be present. These cases are frequent in patients in whom the history shows mouth breathing or thumb sucking as predominant factors. Owing mainly to the non-occlusion present in the anterior region, an afunctional gingivitis, followed by a "horizontal pyorrhea," will invariably take place unless correction of the bite or artificial stimulation is resorted to. In these cases orthodontia is indicated if the age will allow.

Acquired cases arise from accident, abnormal habits, the use of appliances (pipe, etc.), or faulty restorative work in the mouth. I have seen many cases in which the use of restorations higher than the line of occlusion on one or more teeth had caused not only a traumatic occlusion on the supporting teeth, but also a condition of non-occlusion

FIG. 1

FIG. 2

FIG. 1. Non-occlusion on anterior teeth (artichoke bite), with hypoplasia of enamel (Hutchinson's teeth).

FIG. 2. Non-occlusion on anterior teeth. No interference with enamel formation

on the remaining teeth, with disastrous sequelae. I have noticed a condition in men in which the bite on the anteriors is opened, many times quite markedly, the space gradually lessening posteriorly, because the jaws were held apart by a pipe between the teeth for long periods while the third molars were erupting. The third molars were allowed to assume a greater height than was necessary to reach the line of occlusion, resulting in an opening of the bite on the other teeth (*fig. 3*). In these cases correction can very often be accomplished by grinding on the third molars. If these have erupted too markedly, extraction of the offending teeth is obligatory in order to restore proper functional occlusion.

The constant presence in the mouth of a pipe stem (especially a clay pipe), cigar, or cigarette holder has been observed to cause localized non-occlusion when the position of the appliance in the mouth

FIG. 3. Bite held off in protrusion by third molars. This case was not due to pipe smoking, but to eruption of the third molars in centric balance. *A*—centric. *B*—protrusive.

FIG. 4A

FIG. 4B

FIG. 4A. Centric position, showing overjet

FIG. 4B. Same case in protrusive position, showing lack of contact. This case could be corrected by grinding.

is fairly constant. Non-occlusion in this case is due either to the wearing away of the morsal surfaces of the teeth themselves (*fig. 5*), or to an actual depression of the teeth in their sockets resulting in a space between the biting surfaces when the pipe is not in position

(*fig. 6*). As long as the habit is maintained the teeth receive their function, and as a rule the condition does not approach dangerous proportions unless a marked lowering of resistance occurs.

Just as the lack of function in any part of the body results in atrophy, lowered local resistance, and disease, so teeth without function become diseased and lost. Nature does not tolerate any tissue or organ that is not functioning, and always attempts to rid itself of anything that is not being used. Muscles that are not given exercise soon become flabby and diminish in size. Organs increase or decrease in size as an added burden or a lessened amount of work is given to them, but

FIG. 5

FIG. 6

FIG. 5. Non-occlusion from wearing away of the enamel due to pipe smoking

FIG. 6. Non-occlusion caused by depression of the teeth in their sockets due to pipe smoking.

atrophy if they become useless. Teeth whose function is absent and not restored are easily affected by disturbing influences, and become ready prey to dental and periodontal disease (Vincent's infection, decay, periodontitis), eventually being lost.

When non-occlusion is due to extraction of opposing teeth with non-replacement, the teeth elongate, become eroded owing to disturbances in the gingiva, and are a ready prey to decay. Nature, in the attempt to give the tooth a restoration of function, often causes a tooth to elongate until the mucous membrane over the opposing alveolar process is met. In this attempt the teeth very often become nuisances both in mastication and speech, and must be extracted. When the

non-occlusion is due to extraction of opposing teeth, it is of the utmost benefit to the patient to replace the opponents as quickly as possible and thereby prevent this migration.

We now come to the treatment of cases in which the teeth in both jaws are present, but because of existing conditions it is impossible to occlude two or more teeth with their antagonists in any position the mandible may assume (*figs. 8, 9, 10*). Our treatment, as in the eradication of any other disease, consists of three steps: first, removal of the cause; second, establishment of health; third, maintenance of health.

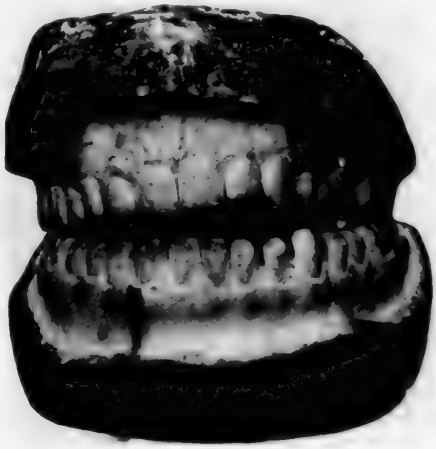

FIG. 7 FIG. 8

FIG. 7. Upper left first bicuspid being pushed into non-occlusion by pipe smoking

FIG. 8. Model of case at beginning of treatment (age 26). Anterior view showing marked non-occlusion. See *figs. 9–11.*

In the treatment of non-occlusion cases, the establishment of healthy circulation by passive massage is of paramount importance. As early as possible the patient should be instructed in the tooth-brush drill, which verily, as Dr. Stillman terms it, is "physical culture for the gums." This passive massage increases the blood supply, hastens the removal of waste materials, and thus, not only raises the resistance of the tissue to disease but also causes a rapid return to health. In this connection I might say that the tooth-brushing massage should

be employed not only on the actual teeth that are in non-occlusion, but upon all the teeth and gingival tissues, in order to increase the resistance of the periodontal structures. It should be continued after actual office treatment has been completed. The passive massage and cleanliness produced by a proper tooth-brush exercise keeps the marginal gingiva healthy and the teeth from eroding during the process of repair.

The next step is to remove deposits, and to curet and treat pockets which may be present. As a rule in non-occlusion cases, the patho-

FIG. 9

FIG. 10

FIG. 9. Same case (*fig. 8*), left side, showing only two molars in contact
FIG. 10. Same case (*figs. 8* and *9*), right side, showing only two molars in contact

logic condition is of the horizontal type, called by Box, periodontitis simplex, and the pockets are found to be shallow. Deep curetting, therefore, is seldom necessary. The teeth should be kept as clean as possible, and tooth-brush massage faithfully carried out for at least ten minutes twice a day during the time the teeth are not in contact.

We are now confronted with the problem of bringing the teeth into occlusion. Three roads are available out of the difficulty: grinding, orthodontia, and active massage. The average case necessitates resort to at least two if not all three of these factors. The grinding

and active massage come under the supervision of the periodontist and in many cases suffice to bring the teeth back to the functional relation at the same time that health is being restored.

The method of grinding for the relief of non-occlusion is as a rule, not complicated and, if the distance between the non-occluding surfaces is not too great, suffices to bring the teeth into function. The patient is instructed to bite on thick Rudischauser articulating paper, and the teeth which strike are then noted. Grinding is done with Chayes stones #5 and #64. The spots marked heaviest should be ground on both upper and lower teeth. Again articulating paper is inserted, the patient is instructed to glide and the new markings are

FIG. 11

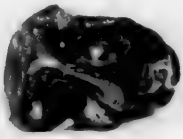

FIG. 12

FIG. 11. Same patient (*figs. 8–10*), after all posteriors were brought into contact by grinding. See *fig.* 14.

FIG. 12. Bite block.

ground. In this way we can remove the high spots evenly throughout the mouth and bring the non-occluding teeth into position without disturbing the balance greatly at any time. Too much grinding should not be done at once. It is rather advisable to divide the period of grinding into several visits. This will give the patient more comfort and will allow him to become accustomed to the new bite.

When it is seen that the amount of grinding needed to bring the non-occluding teeth into contact is so great that it will noticeably close the "degree of opening," or that too great an amount of tooth structure will be sacrificed in the attempt to correct the condition, active biting exercise is indicated (*fig. 11*). We here work on the principle of stimulating growth in the alveolar process by an active

massage that would coincide in its effects in some way with the exercise or stimulation received by the periodontal tissues in mastication. This stimulation is accomplished by an elastic or semi-soft biting block (*figs. 12, 13*), which is so constructed as to conform to the shape of the lower and upper teeth to be stimulated and will, when pressure is not applied, cause the teeth to be held apart to a greater degree than ordinarily. Pressure upon the block to an extent sufficient to cause the naturally occluding teeth to meet would necessitate a stimulating force to be applied to the teeth in the bite block. We thus have a means of evenly stimulating all the teeth which are in non-occlusion, can regulate the amount of pressure to be applied, and can prevent irritation to the peridontal tissues.

FIG. 13

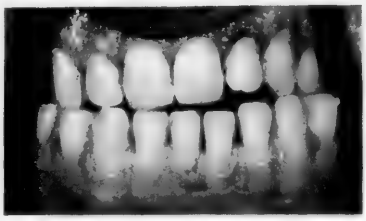

FIG. 14

FIG. 13. Bite block in position. See *fig. 14.*

FIG. 14. Same case (*fig. 11*) after two months of bite-block stimulation, showing all teeth in contact except left central and lateral incisors.

The bite block is constructed as follows: With the bite opened slightly (either before any grinding is done on the naturally occluding teeth, or with a layer of modelling compound or base-plate gutta-percha about 1.5 mm. thick placed in the molar region), a wax bite is taken of the teeth to be stimulated. This should extend well onto the teeth, but not over the gingiva. About two-thirds of the clinical crown should be covered. This can now be carved and shaped and tried in the mouth without stays on the posterior teeth, so as to check the amount of pressure that will finally be applied. The bite is now invested, the wax boiled out, and the mould packed with velum rubber and vulcanized. Only slight trimming on the vulcanized appliance is necessary. The bite block can now be laid aside until the necessary grinding on the occluding teeth and prophylactic treatment

have been accomplished, and tooth-brush massage is well under way. When all this has been done, the patient is instructed in the use of the bite block and given the appliance for home use. The bite block should be placed in the mouth in such a position that the teeth to be stimulated fit into it properly. Sufficient pressure is applied to occlude the ordinarily occluding teeth. This is repeated ten times, morning and evening the first week; fifteen times twice a day, the second week; twenty times twice a day, the third week; and then, continuing at twenty-five times twice a day. The patient should be seen about once a week to note that no injury is being produced by over stimulation.

The condition, originally due to an impairment of bone growth in the alveolar tissue of the non-occluding area, frequently responds to this stimulation and a gradual growth, with movement of the opposing teeth toward each other, occurs. The closing of the bite is not due to lengthening of the clinical crown, but is caused, as may be seen in *figs. 11* and *14*, by a growth in the alveolar process itself. The active stimulation also increases the health and tone of the gingival tissues, and thus aids the passive massage accomplished with the tooth brush.

While teeth are being brought down into occlusion, the bite block should be altered to remove the artificial stimulation of these teeth as natural stimulation comes into play. Tooth brushing, on the other hand, should be continued throughout.

Although I have experienced very gratifying results with this method of treatment, success is greatly dependent upon the particular reaction of the individual case.

Orthodontia should be resorted to when deemed the most appropriate form of treatment for the particular case, and may very often be used with this method to decided advantage.

Let us hope that, by following this simplified technique, we can establish health and maintain it as well as we have learned to do through the teachings of Drs. McCall and Stillman in cases originating in traumatic occlusion.

I wish to acknowledge my thanks to Dr. McCall for his advice and aid in this work.

UNIV. OF MICH.

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By

WILLIAM K. GREGORY, A.M., Ph.D.

Curator of the Department of Comparative Anatomy, American Museum of Natural History, New York; Associate Professor of Vertebrate Palæontology, Columbia University

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